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DATE: Tuesday, November 05, 2002

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L3	514/1.ccls	0	L3
L2	Gli1	18	L2
L1	Altaba-A\$.in.	2	L1

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NEWS 8 Apr 22 Federal Research in Progress (FEDRIP) now available
NEWS 9 Jun 03 New e-mail delivery for search results now available
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NEWS 11 Jun 10 PCTFULL has been reloaded
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NEWS 15 Jul 30 NETFIRST to be removed from STN
NEWS 16 Aug 08 CANCERLIT reload
NEWS 17 Aug 08 PHARMAMarketLetter(PHARMAML) - new on STN
NEWS 18 Aug 08 NTIS has been reloaded and enhanced
NEWS 19 Aug 19 Aquatic Toxicity Information Retrieval (AQUIRE)
                 now available on STN
NEWS 20 Aug 19 IFIPAT, IFICDB, and IFIUDB have been reloaded
NEWS 21 Aug 19 The MEDLINE file segment of TOXCENTER has been reloaded
NEWS 22 Aug 26 Sequence searching in REGISTRY enhanced
NEWS 23 Sep 03 JAPIO has been reloaded and enhanced
NEWS 24 Sep 16 Experimental properties added to the REGISTRY file
NEWS 25 Sep 16 Indexing added to some pre-1967 records in CA/CAPLUS
NEWS 26 Sep 16 CA Section Thesaurus available in CAPLUS and CA
NEWS 27 Oct 01 CASREACT Enriched with Reactions from 1907 to 1985
NEWS 28 Oct 21 EVENTLINE has been reloaded
NEWS 29 Oct 24 BEILSTEIN adds new search fields
NEWS 30 Oct 24 Nutraceuticals International (NUTRACEUT) now available on STN
NEWS 31 Oct 25 MEDLINE SDI run of October 8, 2002
NEWS EXPRESS October 14 CURRENT WINDOWS VERSION IS V6.01,
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              AND CURRENT DISCOVER FILE IS DATED 01 OCTOBER 2002
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FILE 'MEDLINE' ENTERED AT 14:55:36 ON 05 NOV 2002

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=> s altaba-a?/au

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=> s Glil

367 GLI1 1.2

=> s 11 and 12

12 L1 AND L2 L3

=> dup rem 13

PROCESSING COMPLETED FOR L3

10 DUP REM L3 (2 DUPLICATES REMOVED)

=> d ibib abs 1-10

L4 ANSWER 1 OF 10 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.

Full Text

ACCESSION NUMBER: DOCUMENT NUMBER:

2001:530674 BIOSIS

PREV200100530674

TITLE:

Methods and materials for the diagnosis and treatment of

sporadic basal cell carcinoma.

AUTHOR(S):

Altaba, Ariel Ruiz i (1)

CORPORATE SOURCE:

(1) New York, NY USA

ASSIGNEE: New York University

PATENT INFORMATION: US 6238876 May 29, 2001

SOURCE:

Official Gazette of the United States Patent and Trademark

Office Patents, (May 29, 2001) Vol. 1246, No. 5, pp. No

Pagination. e-file.

ISSN: 0098-1133.

DOCUMENT TYPE:

Patent

LANGUAGE:

English

Methods for detection of the onset or presence of sporadic basal cell carcinoma in an animal by measuring for elevated levels of ectopic expression of Gli1 in the animal's epidermal tissue sample suspected of harboring sporadic basal cell carcinoma.

L4 ANSWER 2 OF 10 SCISEARCH COPYRIGHT 2002 ISI (R)

Full Text

ACCESSION NUMBER:

2002:88822 SCISEARCH

THE GENUINE ARTICLE: 514JC

TITLE:

The Sonic Hedgehog-Gli pathway regulates dorsal brain

growth and tumorigenesis

Dahmane N; Sanchez P; Gitton Y; Palma V; Sun T; Beyna M; AUTHOR:

Weiner H; Altaba A R I (Reprint)

NYU, Sch Med, Skirball Inst Biomol Med, Dev Genet Program, CORPORATE SOURCE:

> 540 1st Ave, New York, NY 10016 USA (Reprint); NYU, Sch Med, Skirball Inst Biomol Med, Dev Genet Program, New York, NY 10016 USA; NYU, Sch Med, Dept Cell Biol, New York, NY 10016 USA; NYU, Sch Med, Dept Neurosurg, New

York, NY 10016 USA

COUNTRY OF AUTHOR:

SOURCE:

DEVELOPMENT, (DEC 2001) Vol. 128, No. 24, pp. 5201-5212. Publisher: COMPANY OF BIOLOGISTS LTD, BIDDER BUILDING

CAMBRIDGE COMMERCIAL PARK COWLEY RD, CAMBRIDGE CB4 4DL,

CAMBS, ENGLAND. ISSN: 0950-1991.

DOCUMENT TYPE:

Article; Journal

LANGUAGE:

English

REFERENCE COUNT: 73

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

The mechanisms that regulate the growth of the brain remain unclear. We show that Sonic hedgehog (Shh) is expressed in a layer-specific manner in the perinatal mouse neocortex and tectum, whereas the Gli genes, which are targets and mediators of SHH signaling, are expressed in proliferative zones. In vitro and in vivo assays show that SHH is a mitogen for neocortical and tectal precursors and that it modulates cell proliferation in the dorsal brain. Together with its role in the cerebellum, our findings indicate that SHH signaling unexpectedly controls the development of the three major dorsal brain structures. We also show that a variety of primary human brain tumors and tumor lines consistently express the GLI genes and that cyclopamine, a SHH signaling inhibitor, inhibits the proliferation of tumor cells. Using the in vivo tadpole assay system, we further show that misexpression of GLI1 induces CNS hyperproliferation that depends on the activation of endogenous Gli1 function. SHH-GLI signaling thus modulates normal dorsal brain growth by controlling precursor proliferation, an evolutionarily important and plastic process that is deregulated in brain tumors.

ANSWER 3 OF 10 SCISEARCH COPYRIGHT 2002 ISI (R) L4

Full Text

ACCESSION NUMBER: 2001:424370 SCISEARCH

THE GENUINE ARTICLE: 433NA

TITLE:

Wnt signals are targets and mediators of Gli function AUTHOR: Mullor J L; Dahmane N; Sun T; Altaba A R I (Reprint)

NYU, Sch Med, Dev Genet Program, Skirball Inst, 540 1st CORPORATE SOURCE: Ave, New York, NY 10016 USA (Reprint); NYU, Sch Med, Dev

Genet Program, Skirball Inst, New York, NY 10016 USA; NYU,

Sch Med, Dept Cell Biol, New York, NY 10016 USA

COUNTRY OF AUTHOR: USA

SOURCE:

CURRENT BIOLOGY, (15 MAY 2001) Vol. 11, No. 10, pp.

769-773.

Publisher: CELL PRESS, 1100 MASSACHUSETTES AVE,,

CAMBRIDGE, MA 02138 USA.

ISSN: 0960-9822. Article; Journal

DOCUMENT TYPE: LANGUAGE:

English

REFERENCE COUNT:

30

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

There is growing evidence that Gli proteins participate in the mediation of Hedgehog and FGF signaling in neural and mesodermal development. However, little is known about which genes act downstream of Gli proteins, Here we show the regulation of members of the Wnt family by Gli proteins in different contexts. Our findings indicate that Gli2

regulates Wnt8 expression in the ventral marginal zone of the early frog embryo: activating Gli2 constructs induce ectopic Wnt8 expression in animal cap explants, whereas repressor forms inhibit its endogenous expression in the marginal zone. Using truncated Frizzled and dominant-negative Wnt constructs, we then show the requirement of at least two Wnt proteins, Wnt8 and Wnt11, for Gli2/3-induced posterior mesodermal development. Blocking Wnt signals, however, inhibits Gli2/3-induced morphogenesis, but not mesodermal specification, Gli2/3 may therefore normally coordinate the action of these two Wnt proteins, which regulate distinct downstream pathways. In addition, the finding that Gli1 consistently induces a distinct set of Wnt genes in animal cap explants and in skin tumors suggests that Wnt regulation by Gli proteins is general, Such a mechanism may link signals that induce Gli activity, such as pops and Hedgehogs, with Wnt function.

L4 ANSWER 4 OF 10 SCISEARCH COPYRIGHT 2002 ISI (R)

Full Text

ACCESSION NUMBER: 2000:862496 SCISEARCH

THE GENUINE ARTICLE: 372KF

TITLE: Gli2 functions in FGF signaling during antero-posterior

patterning

AUTHOR: Brewster R; Mullor J L; Altaba A R (Reprint)

CORPORATE SOURCE: NYU, SCH MED, SKIRBALL INST, DEV GENET PROGRAM, NEW YORK,

NY 10016 (Reprint); NYU, SCH MED, SKIRBALL INST, DEV GENET PROGRAM, NEW YORK, NY 10016; NYU, SCH MED, DEPT CELL BIOL,

NEW YORK, NY 10016

COUNTRY OF AUTHOR: USA

SOURCE: DEVELOPMENT, (OCT 2000) Vol. 127, No. 20, pp. 4395-4405.

Publisher: COMPANY OF BIOLOGISTS LTD, BIDDER BUILDING CAMBRIDGE COMMERCIAL PARK COWLEY RD, CAMBRIDGE CB4 4DL,

CAMBS, ENGLAND. ISSN: 0950-1991. Article; Journal

FILE SEGMENT: LIFE LANGUAGE: English

REFERENCE COUNT: 81

DOCUMENT TYPE:

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

Patterning along the anteroposterior (A-P) axis involves the interplay of secreted and transcription factors that specify cell fates in the mesoderm and neuroectoderm. While FGF and homeodomain proteins have been shown to play different roles in posterior specification, the network coordinating their effects remains elusive, Here we have analyzed the function of Gli zinc-finger proteins in mesodermal A-P patterning. We find that Gli2 is sufficient to induce ventroposterior development, functioning in the FGF-brachyury regulatory loop. Gli2 directly induces brachyury, a gene required and sufficient for mesodermal development, and Gli2 is in turn induced by FGF signaling. Moreover, the homeobox gene Xhox3, a critical determinant of posterior development, is also directly regulated by Gli2. Gli3, but not Gli1, has an activity similar to that of Gli2 and is expressed in ventroposterior mesoderm after Gli2, These findings uncover a novel function of Gli proteins, previously only known to mediate hedgehog signals, in the maintenance and patterning of the embryonic mesoderm. More generally, our results suggest a molecular basis for an integration of FGF and hedgehog inputs in Gli-expressing cells that respond to these signals.

L4 ANSWER 5 OF 10 SCISEARCH COPYRIGHT 2002 ISI (R)

Full Text

ACCESSION NUMBER: 2000:846783 SCISEARCH

THE GENUINE ARTICLE: 370KH

TITLE: Expression of the vertebrate Gli proteins in Drosophila

reveals a distribution of activator and repressor

activities

AzaBlanc P; Lin H Y; Altaba A R I; Kornberg T B (Reprint) AUTHOR:

CORPORATE SOURCE: UNIV CALIF SAN FRANCISCO, DEPT BIOCHEM BIOPHYS, SAN

> FRANCISCO, CA 94143 (Reprint); UNIV CALIF SAN FRANCISCO, DEPT BIOCHEM BIOPHYS, SAN FRANCISCO, CA 94143; NYU, SCH MED, DEPT CELL BIOL, NEW YORK, NY 10016; NYU, SKIRBALL

INST, DEV GENET PROGRAM, NEW YORK, NY 10016

COUNTRY OF AUTHOR:

SOURCE: DEVELOPMENT, (OCT 2000) Vol. 127, No. 19, pp. 4293-4301.

> Publisher: COMPANY OF BIOLOGISTS LTD, BIDDER BUILDING CAMBRIDGE COMMERCIAL PARK COWLEY RD, CAMBRIDGE CB4 4DL,

CAMBS, ENGLAND. ISSN: 0950-1991.

DOCUMENT TYPE: Article; Journal

FILE SEGMENT: LIFE LANGUAGE: English REFERENCE COUNT: 44

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AΒ The Cubitus interruptus (Ci) and Gli proteins are transcription factors that mediate responses to Hedgehog proteins (Hh) in flies and vertebrates, respectively. During development of the Drosophila wing, Ci transduces the Hh signal and regulates transcription of different target genes at different locations. In vertebrates, the three Gli proteins are expressed in overlapping domains and are partially redundant. To assess how the vertebrate Glis correlate with Drosophila Ci, we expressed each in Drosophila and monitored their behaviors and activities. We found that each Gli has distinct activities that are equivalent to portions of the regulatory arsenal of Ci. Gli2 and Gli1 have activator functions that depend on Hh, Gli2 and Gli3 are proteolyzed to produce a repressor form able to inhibit hh expression. However, while Gli3 repressor activity is regulated by Hh, Gli2 repressor activity is not. These observations suggest that the separate activator and repressor functions of Ci are unevenly partitioned among the three Glis, yielding proteins with related yet distinct properties.

L4ANSWER 6 OF 10 SCISEARCH COPYRIGHT 2002 ISI (R)

Full Text

ACCESSION NUMBER: 1999:616705 SCISEARCH

THE GENUINE ARTICLE: 223BA

TITLE: Gli proteins encode context-dependent positive and

negative functions: implications for development and

disease

AUTHOR: Altaba A R I (Reprint)

NYU, SCH MED, SKIRBALL INST, DEV GENET PROGRAM, 540 1ST CORPORATE SOURCE:

AVE, NEW YORK, NY 10016 (Reprint); NYU, SCH MED, DEPT CELL

BIOL, NEW YORK, NY 10016

COUNTRY OF AUTHOR: USA

SOURCE: DEVELOPMENT, (JUL 1999) Vol. 126, No. 14, pp. 3205-3216.

> Publisher: COMPANY OF BIOLOGISTS LTD, BIDDER BUILDING CAMBRIDGE COMMERCIAL PARK COWLEY RD, CAMBRIDGE CB4 4DL,

CAMBS, ENGLAND. ISSN: 0950-1991. Article: Journal

FILE SEGMENT: LIFE

DOCUMENT TYPE:

LANGUAGE: English REFERENCE COUNT: 60

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

Several lines of evidence implicate zinc finger proteins of the Gli family in the final steps of Hedgehog signaling in normal development and disease, C-terminally truncated mutant GLI3 proteins are also associated with human syndromes, but it is not clear whether these C-terminally truncated Gli proteins fulfil the same function as full-length ones. Here,

structure-function analyses of Gli proteins have been performed using floor plate and neuronal induction assays in frog embryos, as well as induction of alkaline phosphatase (AP) in SHH-responsive mouse C3H10T1/2 (10T1/2) cells. These assays show that C-terminal sequences are required for positive inducing activity and cytoplasmic Localization, whereas N-terminal sequences determine dominant negative function and nuclear localization. Analyses of nuclear targeted Gli1 and Gli2 proteins suggest that both activator and dominant negative proteins are modified forms. In embryos and COS cells, tagged Gli cDNAs yield C-terminally deleted forms similar to that of Ci, These results thus provide a molecular basis for the human Polydactyly type A and Pallister-Hall Syndrome phenotypes, derived from the deregulated production of C-terminally truncated GLI3 proteins. Analyses of full-length Gli function in 10T1/2 cells suggest that nuclear localization of activating forms is a regulated event and show that only Glil mimics SHH in inducing AP activity.: Moreover, full-length Gli3 and all C-terminally truncated forms act antagonistically whereas Gli2 is inactive in this assay. In 10T1/2 cells, protein kinase A (PKA), a known inhibitor of Hh signaling, promotes Gli3 repressor formation and inhibits Gli1 function. Together, these findings suggest a context-dependent functional divergence of Gli protein function, in which a cell represses Gli3 and activates Cli1/2 prevents the formation of repressor Gli forms to respond to Shh. Interpretation of Hh signals by Gli proteins therefore appears to involve a fine balance of divergent functions within each and among different Gli proteins, the misregulation of which has profound biological consequences.

L4 ANSWER 7 OF 10 SCISEARCH COPYRIGHT 2002 ISI (R)

Full Text

ACCESSION NUMBER: 1998:542482 SCISEARCH

THE GENUINE ARTICLE: ZZ232

TITLE: Combinatorial Gli gene function in floor plate and

neuronal inductions by sonic hedgehog

AUTHOR: Altaba A R I (Reprint)

CORPORATE SOURCE: NYU, MED CTR, SKIRBALL INST, DEV GENET PROGRAM, 540 1ST

AVE, NEW YORK, NY 10016 (Reprint); NYU, MED CTR, DEPT CELL

BIOL, NEW YORK, NY 10016

COUNTRY OF AUTHOR: USA

SOURCE: DEVELOPMENT, (JUN 1998) Vol. 125, No. 12, pp. 2203-2212.

Publisher: COMPANY OF BIOLOGISTS LTD, BIDDER BUILDING CAMBRIDGE COMMERCIAL PARK COWLEY RD, CAMBRIDGE CB4 4DL,

CAMBS, ENGLAND. ISSN: 0950-1991. Article; Journal

FILE SEGMENT: LIFE LANGUAGE: English

DOCUMENT TYPE:

REFERENCE COUNT:

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

Within the developing vertebrate nervous system, it is not known how AB progenitor cells interpret the positional information provided by inducing signals or how the domains in which distinct groups of neural cells differentiate are defined. Gli proteins may be involved in these processes. In the frog neural plate, we have previously shown that the zinc finger transcription factor Gli1 is expressed in midline cells and mediates the effects of Shh inducing door plate differentiation. In contrast, Gli2 and Gli3 are expressed throughout the neural plate except for the midline. Here, it is shown that Gli3 and Shh repress each other whereas Gli2, like Gli1, is a target of Shh signaling. However, only Gli1 can induce the differentiation of boor plate cells. In addition, Gli2 and Gli3 repress the ectopic induction of floor plate cells by Gli1 in co-injection assays and inhibit endogenous floor plate differentiation, The definition of the boor plate domain, therefore, appears to be defined by the antagonizing activities of Gli2 and Gli3 on Gli1 function.

Because both **Glil** and Glil are induced by Shh, these results establish a regulatory feedback loop triggered by Shh that restricts floor plate cells to the midline. We have also previously shown that the Gli genes induce neuronal differentiation and here it is shown that there is specificity to the types of neurons the Oh proteins induce. Only **Glil** induces Nkx2.1/TTF-1(+) ventral forebrain neurons. Moreover, Glil and Glil inhibit their differentiation. In contrast, the differentiation of spinal motor neurons can be induced by the two ventrally expressed Gli genes, **Glil** and Glil, suggesting that Glil directly mediates induction of motor neurons by Shh. In addition, Glil inhibits motor neuron differentiation by Glil. Thus, combinatorial Gli function may pattern the neural tube, integrating positional information and cell type differentiation.

L4 ANSWER 8 OF 10 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.DUPLICATE 1

Full Text

ACCESSION NUMBER: 97245195 EMBASE

DOCUMENT NUMBER: 1997245195

TITLE: Gli1 is a target of Sonic hedgehog that induces ventral

neural tube development.

AUTHOR: Lee J.; Platt K.A.; Censullo P.; Altaba A.R.I.

CORPORATE SOURCE: A.R.I. Altaba, The Skirball Institute, Developmental

Genetics Program, NYU Medical Center, 540 First Avenue, New

York, NY 10016, United States. ria@saturn.med.nvu.edu

SOURCE: Development, (1997) 124/13 (2537-2552).

Refs: 112

ISSN: 0950-1991 CODEN: DEVPED

COUNTRY: United Kingdom DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 021 Developmental Biology and Teratology

022 Human Genetics

LANGUAGE: English
SUMMARY LANGUAGE: English

The vertebrate zinc finger genes of the Gli family are homologs of the Drosophila gene cubitus interruptus. In frog embryos, Glil is expressed transiently in the prospective floor plate during gastrulation and in cells lateral to the midline during late gastrula and neurula stages. In contrast, Gli2 and Gli3 are absent from the neural plate midline with Gli2 expressed widely and Gli3 in a graded fashion with highest levels in lateral regions. In mouse embryos, the three Gli genes show a similar pattern of expression in the neural tube but are coexpressed throughout the early neural plate. Because Gli1 is the only Gli gene expressed in prospective door plate cells of frog embryos, we have investigated a possible involvement of this gene in ventral neural tube development. Here we show that Shh signaling activates Gli1 transcription and that widespread expression of endogenous frog or human glioma Gli1, but not Gli3, in developing frog embryos results in the ectopic differentiation of floor plate cells and ventral neurons within the neural tube. Floor-plate-inducing ability is retained when cytoplasmic Gli1 proteins are forced into the nucleus or are fused to the VP16 transactivating domain. Thus, our results identify Gli1 as a midline target of Shh and suggest that it mediates the induction of floor plate cells and ventral neurons by Shh acting as a transcriptional regulator.

L4 ANSWER 9 OF 10 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.DUPLICATE 2

Full Text

ACCESSION NUMBER: 97332893 EMBASE

DOCUMENT NUMBER: 1997332893

TITLE: Activation of the transcription factor Gli1 and the sonic

hedgehog signalling pathway in skin tumours.

AUTHOR: Dahmane N.; Lee J.; Robins P.; Heller P.; Altaba A.R.

CORPORATE SOURCE: A.R. Altaba, Skirball Institute, Developmental Genetics

Program, New York University Medical Center, 540 First

Avenue, New York, NY 10016, United States.

ria@saturn.med.nvu.edu

SOURCE: Nature, (19

Nature, (1997) 389/6653 (876-881). O

Refs: 29

ISSN: 0028-0836 CODEN: NATUAS

COUNTRY:
DOCUMENT TYPE:
FILE SEGMENT:

United Kingdom
Journal; Article
Ol6 Cancer

022 Human Genetics

LANGUAGE:

English

SUMMARY LANGUAGE:

English

Sporadic basal cell carcinoma (BCC) is the most common type of malignant cancer in fair-skinned adults. Familial BCCs and a fraction of sporadic BCCs have lost the function of Patched (Ptc), a Sonic hedgehog (Shh) receptor that acts negatively on this signalling pathway. Overexpression of shh can induce BCCs in mice. Here we show that ectopic expression of the zinc-finger transcription factor Gli1 in the embryonic frog epidermis results in the development of tumours that express endogenous Gli1. We also show that Shh and the Gli genes are normally expressed in hair follicles, and that human sporadic BCCs consistently express Gli1 but not Shh or Gli3. Because Gli1, but not Gli3, acts as a target and mediator of Shh signalling, our results suggest that expression of Gill in basal cells induces BCG formation. Moreover, loss of Ptc or overexpression of Shh cannot be the sole causes of Gill induction and sporadic BCC formation, as they do not occur consistently. Thus any mutations leading to the expression of GIll in basal cells are predicted to induce BCC formation.

L4 ANSWER 10 OF 10 SCISEARCH COPYRIGHT 2002 ISI (R)

Full Text

ACCESSION NUMBER: 97:891461 SCISEARCH

THE GENUINE ARTICLE: YJ865

TITLE: Activation of the transcription factor Gli1 and the

Sonic hedgehog signalling pathway in skin tumours (vol

389, pg 876, 1997)

AUTHOR:

Dahmane N (Reprint); Lee J; Robins P; Heller P; Altaba A

RI

SOURCE:

NATURE, (4 DEC 1997) Vol. 390, No. 6659, pp. 536-536. Publisher: MACMILLAN MAGAZINES LTD, PORTERS SOUTH, 4

CRINAN ST, LONDON, ENGLAND N1 9XW.

ISSN: 0028-0836. Errata; Journal

DOCUMENT TYPE: FILE SEGMENT:

PHYS; LIFE; AGRI

LANGUAGE:

English

REFERENCE COUNT:

=> d his

(FILE 'HOME' ENTERED AT 14:55:14 ON 05 NOV 2002)

FILE 'MEDLINE, CANCERLIT, BIOSIS, EMBASE, SCISEARCH' ENTERED AT 14:55:36 ON 05 NOV 2002

L1 70 S ALTABA-A?/AU

L2 367 S GLI1

L3 12 S L1 AND L2

L4 10 DUP REM L3 (2 DUPLICATES REMOVED)

=> s 12 and py<=1997

2 FILES SEARCHED...

3 FILES SEARCHED...

L5 18 L2 AND PY<=1997

=> dup rem 15

PROCESSING COMPLETED FOR L5

L6 8 DUP REM L5 (10 DUPLICATES REMOVED)

=> d ibib abs 1-8

L6 ANSWER 1 OF 8 MEDLINE DUPLICATE 1

Full Text

ACCESSION NUMBER: 97359968 MEDLINE

DOCUMENT NUMBER: 97359968 PubMed ID: 9216996

TITLE: Gli1 is a target of Sonic hedgehog that induces ventral

neural tube development.

AUTHOR: Lee J; Platt K A; Censullo P; Ruiz i Altaba A

CORPORATE SOURCE: The Skirball Institute, Developmental Genetics Program and

Department of Cell Biology, NYU Medical Center, New York,

NY 10016, USA.

SOURCE: DEVELOPMENT, (1997 Jul) 124 (13) 2537-52.

Journal code: 8701744. ISSN: 0950-1991.

PUB. COUNTRY: ENGLAND: United Kingdom

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals OTHER SOURCE: GENBANK-U57454

ENTRY MONTH: 199708

ENTRY DATE: Entered STN: 19970813

Last Updated on STN: 19970813 Entered Medline: 19970807

The vertebrate zinc finger genes of the Gli family are homologs of the Drosophila gene cubitus interruptus. In frog embryos, Glil is expressed transiently in the prospective floor plate during gastrulation and in cells lateral to the midline during late gastrula and neurula stages. In contrast, Gli2 and Gli3 are absent from the neural plate midline with Gli2 expressed widely and Gli3 in a graded fashion with highest levels in lateral regions. In mouse embryos, the three Gli genes show a similar pattern of expression in the neural tube but are coexpressed throughout the early neural plate. Because Gli1 is the only Gli gene expressed in prospective floor plate cells of frog embryos, we have investigated a possible involvement of this gene in ventral neural tube development. Here we show that Shh signaling activates Gli1 transcription and that widespread expression of endogenous frog or human glioma Gli1, but not Gli3, in developing frog embryos results in the ectopic differentiation of floor plate cells and ventral neurons within the neural tube. Floor-plate-inducing ability is retained when cytoplasmic Gli1 proteins are forced into the nucleus or are fused to the VP16 transactivating domain. Thus, our results identify Glil as a midline target of Shh and suggest that it mediates the induction of floor plate cells and ventral neurons by Shh acting as a transcriptional regulator.

L6 ANSWER 2 OF 8 MEDLINE DUPLICATE 2

Full Text

ACCESSION NUMBER: 1998007978 MEDLINE

DOCUMENT NUMBER: 98007978 PubMed ID: 9349822

TITLE: Activation of the transcription factor Gli1 and the Sonic

hedgehog signalling pathway in skin tumours.

COMMENT: Erratum in: Nature 1997 Dec 4;390(6659):536

AUTHOR: Dahmane N; Lee J; Robins P; Heller P; Ruiz i Altaba A CORPORATE SOURCE: The Skirball Institute, Department of Cell Biology, New

York University Medical Center, New York 10016, USA.

SOURCE: NATURE, (1997 Oct 23) 389 (6653) 876-81.

Journal code: 0410462. ISSN: 0028-0836.

PUB. COUNTRY: ENGLAND: United Kingdom

DOCUMENT TYPE: Journal: Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 199711

ENTRY DATE: Entered STN: 19971224

Last Updated on STN: 19990129 Entered Medline: 19971113

Sporadic basal cell carcinoma (BCC) is the most common type of malignant cancer in fair-skinned adults. Familial BCCs and a fraction of sporadic BCCs have lost the function of Patched (Ptc), a Sonic hedgehog (Shh) receptor that acts negatively on this signalling pathway. Overexpression of Shh can induce BCCs in mice. Here we show that ectopic expression of the zinc-finger transcription factor Gli1 in the embryonic froq epidermis results in the development of tumours that express endogenous Gli1. We also show that Shh and the Gli genes are normally expressed in hair follicles, and that human sporadic BCCs consistently express Gli1 but not Shh or Gli3. Because Gli1, but not Gli3, acts as a target and mediator of Shh signalling, our results suggest that expression of Gli1 in basal cells induces BCC formation. Moreover, loss of Ptc or overexpression of Shh cannot be the sole causes of Gli1 induction and sporadic BCC formation, as they do not occur consistently. Thus any mutations leading to the expression of Gli1 in basal cells are predicted to induce BCC formation.

L6 ANSWER 3 OF 8 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.

Full Text

ACCESSION NUMBER: 1998:121175 BIOSIS
DOCUMENT NUMBER: PREV199800121175

TITLE: Correction of PREVIEWS 99816116. Activation of the

transcription factor **Gli1** and the sonic hedgehog signalling pathway in skin tumours. Replacement of abstract. Erratum published in Nature (London) Vol. 390.

Iss. 6659. 1997. p. 536.

AUTHOR(S): Dahmane, N.; Lee, J.; Robins, P.; Heller, P.; Ruiz I

Altaba, A. (1)

CORPORATE SOURCE: (1) Skirball Inst., Dev. Genet. Program, 540 First Ave.,

New York, NY 10016 USA

SOURCE: Nature (London), (Dec. 4, 1997) Vol. 390, No. 6659, pp.

876-881.

ISSN: 0028-0836.

DOCUMENT TYPE: Article; Errata

LANGUAGE: English

Patients with basal cell nevus syndrome develop basal cell carcinomas (BCCs) early in life and carry mutations in the Patched gene, which encodes a receptor for the Sonic hedgehog ligand. These findings implicated the activation of the Sonic hedgehog signalling pathway in the familial or inherited form of BCC. However, the molecular mechanisms underlying the development of sporadic BCCs, the commonest form of skin cancer in fair-skinned adults with over a million cases a year worldwide, remained unknown. Now Dahmane et al. provide compelling evidence that virtually all sporadic BCCs have the Shh signalling pathway activated as determined by the expression of the zinc finger transcription factor Gli1, the final target and mediator of Shh signalling. The work predicts that any mutations that lead to the activation of this pathway in basal cells, and thus to Gli1 transcription and function, will cause basal cell cancer. Moreover, work in model organisms shows that inappropriate expression of Gli1 in the skin leads to the development of epidermal tumours. Gli1 may thus be both a marker and cause of BCC formation, making prospects for early diagnosis and possible treatment of this widespread type of skin cancer feasible.

L6 ANSWER 4 OF 8 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.

Full Text

ACCESSION NUMBER:

1998007772 EMBASE

TITLE:

Erratum: Activation of the transcription factor Gli1 and

oct.

the Sonic hedgehog signalling pathway in skin tumours

(Nature (1997) 389 (876-881)).

AUTHOR:

Dahmane N.; Lee J.; Robins P.; Heller P.; Ruiz A.A.

SOURCE:

Nature, (1997) 390/6659 (536). ISSN: 0028-0836 CODEN: NATUAS

COUNTRY:

United Kingdom Journal; Errata

DOCUMENT TYPE: FILE SEGMENT:

016 Cancer

LANGUAGE:

English

L6 ANSWER 5 OF 8 SCISEARCH COPYRIGHT 2002 ISI (R)

Full Text

ACCESSION NUMBER:

97:891461 SCISEARCH

THE GENUINE ARTICLE: YJ865

TITLE:

Activation of the transcription factor Glil and the Sonic hedgehog signalling pathway in skin tumours (vol

389, pg 876, 1997)

AUTHOR:

Dahmane N (Reprint); Lee J; Robins P; Heller P; Altaba A R

SOURCE:

NATURE, (4 DEC 1997) Vol. 390, No. 6659, pp. 536-536. Publisher: MACMILLAN MAGAZINES LTD, PORTERS SOUTH, 4

CRINAN ST, LONDON, ENGLAND N1 9XW.

ISSN: 0028-0836. Errata; Journal

DOCUMENT TYPE: FILE SEGMENT:

PHYS; LIFE; AGRI

LANGUAGE:

English

1

REFERENCE COUNT:

L6 ANSWER 6 OF 8 SCISEARCH COPYRIGHT 2002 ISI (R)

Full Text

ACCESSION NUMBER:

97:617371 SCISEARCH

THE GENUINE ARTICLE: XH774

TITLE:

Functional and molecular characterization of Gli1 in

mouse development

AUTHOR:

Park H L (Reprint); Platt K; Joyner A L

CORPORATE SOURCE:

NYU MED CTR, SKIRBALL INST, NEW YORK, NY 10016

COUNTRY OF AUTHOR:

USA

SOURCE:

DEVELOPMENTAL BIOLOGY, (15 JUN 1997) Vol. 186, No. 2,

pp. B257-B257.

Publisher: ACADEMIC PRESS INC JNL-COMP SUBSCRIPTIONS, 525

B ST, STE 1900, SAN DIEGO, CA 92101-4495.

ISSN: 0012-1606.

DOCUMENT TYPE: FILE SEGMENT:

Conference; Journal LIFE

LANGUAGE:

English

REFERENCE COUNT:

n

L6 ANSWER 7 OF 8 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.

Full Text

ACCESSION NUMBER:

1992:430615 BIOSIS

DOCUMENT NUMBER:

BA94:82740

TITLE:

ANALYSIS OF GLIA CELL DIFFERENTIATION IN THE DEVELOPING CHICK PERIPHERAL NERVOUS SYSTEM SENSORY AND SYMPATHETIC SATELLITE CELLS EXPRESS DIFFERENT CELL SURFACE ANTIGENS.

AUTHOR(S):

RUEDEL C; ROHRER H

CORPORATE SOURCE:

MAX-PLANCK-INST. HIRNFORSCHUNG, ABT. NEUROCHEM.,

DEUTSCHORDENSTRASSE 46, 6000 FRANKFURT/M. 71, GER.

SOURCE:

DEVELOPMENT (CAMB), (1992) 115 (2), 519-526.

CODEN: DEVPED. ISSN: 0950-1991.

FILE SEGMENT: BA; OLD LANGUAGE: English

To identify and analyse precursor cells of neuronal and glial cell lineages during the early development of the chick peripheral nervous system, monoclonal antibodies were raised against a population of undifferentiated cells of E6 dorsal root ganglia (DRG). Non-neuronal cells of E6 DRG express surface antigens that are recognized by four monoclonal antibodies, G1, G2, GLI1 and GLI2. The proportion of non-neuronal cells in DRG that express the GLI 1 antigen is very high during ganglion formation (80% at E4) and decreases during later development (15% at E14). GLI 2 antiqen is expressed only on a minority of the cells at E6 and increases with development. The G1 and G2 antigens are expressed on about 60-80% of the cells between E6 and E14. All cells that express the established glia marker O4 are also positive for the new antigens. In addition, it was demonstrated that GLI 1-positive cells from early DRG, which are devoid of O4 antigen, could be induced in vitro to express the O4 antigen. Thus the antigen-positive cells are considered as glial cells or glial precursor cells. Surprisingly, the antigen expression by satellite cells of peripheral ganglia is dependent on the type of ganglion: antigens G1, G2 and GLI1 were not detectable on glial cells of lumbosacral sympathetic ganglia and GLI2 was expressed only by a small subpopulation. These results demonstrate an early immunological difference between satellite cells of sensory DRG and sympathetic ganglia. As the antigens could however be induced in vitro also in sympathetic gangliion cells, it is susggested that the specific antigen expression is due to specific environmental cues acting on precursor cells in different types of ganglia rather than to intrinsic differences between sensory and sympathetic glial precursor cells.

DUPLICATE 3 ANSWER 8 OF 8 MEDLINE L6

Full Text

ACCESSION NUMBER: 77153474 MEDLINE

PubMed ID: 848131 DOCUMENT NUMBER: 77153474

Population genetics of glyoxalase I (E.C.4.4.1.5) in human TITLE:

erythrocytes.

Berg K; Rodewald A; Schwarzfischer F; Wischerath H AUTHOR:

SOURCE: ZEITSCHRIFT FUR RECHTSMEDIZIN. JOURNAL OF LEGAL MEDICINE,

(1977 Jan 21) 79 (1) 13-5.

Journal code: 0247437. ISSN: 0044-3433. GERMANY, WEST: Germany, Federal Republic of

PUB. COUNTRY: Journal; Article; (JOURNAL ARTICLE) DOCUMENT TYPE:

English LANGUAGE:

Priority Journals FILE SEGMENT:

ENTRY MONTH: 197705

Entered STN: 19900313 ENTRY DATE:

Last Updated on STN: 19980206 Entered Medline: 19770512

1025 individuals from Southern Germany were examined. The gene frequencies AB for GLI1 are 0.4235 and for GLI2 0.5765. These frequencies are compared with those of other authors.

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